pseudoglobulin in the unheated and heated serum divided by the amount of pseudoglobulin in the unheated serum gives the percent transformed; these are tabulated at the bottom of table 2. Thus for Anthrax 48, the figures are $(0.290-0.211)/0.290=27\%$. This is not the only way to calculate this figure. Figures for pseudoglobulin may be obtained from a different set of results; thus, they may be calculated by subtracting the figures for albumin (e) from those or pseudoglobulin plus albumin (c). If the percent of transformation be calculated from these lower values for pseudoglobulin the figures are, 24, 8.4., -1.2, 12.4 and 11.2%; to read across the bottom of table 2.

Further details are given in the *Journal of Agricultural Research*, 1917.

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A CASE OF NORMAL EMBRYONIC ATRESIA OF THE ESOPHAGUS

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In a series of loggerhead turtle embryos, collected and used originally for a study of the history of the primordial germ cells, the esophagus was observed to be solid for a greater or less extent, approximately from the point of origin of the respiratory anlage to its bifurcation into the bronchi, from the twelfth to the thirty-second day of incubation. At the latter stage the esophagus is still occluded at its oral end, though now fenestrated for a considerable extent caudally; and it seems probable that the esophageal atresia persists practically to near the end of the incubation period (eight weeks) at the level just behind the opening of the larynx.

The points of special significance in regard to this material are: (1) the relatively longer persistence of the occlusion than has yet been described for any other form; (2) the absence of contributory yolk in the stenosed area; and (3) close relation of the atresia to the point of origin of the respiratory anlage, which fact may disclose its possible functional significance.

Balfour[1] was the first to describe a similar phenomenon in the esophagus of certain selachii. Kreuter[2, 3] confirmed these observations in the case of *Pristiurus* and *Torpedo*. Dean[4] reports a solid esophagus in larvae of *Amia calva*. An occluded esophagus is said to occur also in certain bony fishes, e.g., herring, trout, salmon (Balfour;

Oppel[5]). In cyclostomes the esophagus remains patent throughout development (Kreuter[3]). In certain amphibia (*Bufo; Rana*) the esophagus becomes occluded, in part through the medium of contributory yolk globules (Meuron[6]); and the same is true for certain reptiles (*Anguis fragilis*, Oppel;[5] *Lacerta*, Meuron[6]). According to Meuron[6] the esophagus of the chick embryo of the fifth day is occluded for a length of 115 microns, but regains partial patency again in the sixth day through the appearance of vacuoles.

Kreuter[3] was the first to describe an epithelial obliteration of the esophageal lumen in the human embryo; contrary to the teaching of Kollmann[7] and other embryologists that no solid stage of the esophagus occurred in mammals and in man. Kreuter[3] describes also similar obliterated areas in the mid- and hind-gut of embryos between the fourth and tenth weeks. In four human embryos, measuring from 8.4 to 16 mm., Lewis[8] describes an esophagus whose lumen is pervious throughout. He, however, describes vacuoles in the epithelial lining of these stages similar to those described by Kreuter as stages in the opening of the solid esophagus. But he regards an atresia of the esophagus in the human embryo as abnormal at all stages (p. 368).

It would seem that an embryonic normal atresia of the esophagus is a widespread phenomenon among vertebrates, and is essentially similar from elasmobranch fishes to man.

The phenomenon has not yet, as far as I am aware, been described for turtles, a circumstance which adds to the interest of this investigation. Nor has its intimate spatial relationship to the respiratory anlage, and its probable functional significance, been hitherto pointed out. The more important results may be summarized as follows:

1. During the tenth and eleventh days of incubation the epithelial lining of the oral end of the esophagus (esophageo-respiratory anlage) thickens greatly dorsally, the result of extensive cell proliferation in this region. During the twelfth day the cylindric tube of the esophagus becomes compressed dorso-ventrally, thus bringing the dorsal and ventral epithelial walls in close apposition. Only the minutest central lumen persists in the oral end of the esophagus for a distance of about 0.25 mm. During the thirteenth day the oral end of the esophagus is rectangular in cross section and completely solid for a distance of about half a millimeter. The opposed central cells have fused and formed a plug of tissue, essentially like a mesenchymal syncytium.

2. The initial point of atresia is *over*, or just behind, the orifice of the separating laryngo-tracheal anlage; and its inception is coincident with the earliest stage in the division of the original esophageo-respira-

tory anlage into an esophageal and a laryngo-tracheal tube. By the sixteenth day the atresia has extended into the orifice of the larynx, due in part perhaps to pressure exerted by the lateral arytenoid swellings.

3. The chief factor in the temporary closure of the originally open esophagus is the change in shape of the esophagus from a tube approximately circular in cross section to a structure of wide rectangular form with at first a slit-like lumen and finally a minute central aperture. The cause of the change in shape, upon which the obliteration of the lumen largely depends, is the combination of growth within the esophagus in opposition to the denser lateral mesenchymal plates, by the invasion and medial fusion of which the laryngo-tracheal groove becomes converted into a tube and incidentally separated from the esophagus distally. This process is assisted, as concerns the obliteration of esophageal lumen, by the active cell proliferation in the dorsal wall of the esophagus.

4. In the sixteen day embryo, the atresia of the esophagus extends through about 1500 microns. Beyond the oral end vacuoles begin to form in the lining epithelium. These represent dilated 'intercellular' spaces chiefly within the central syncytial plug of tissue. They increase in number, and enlarge caudally, where they become confluent. During succeeding stages this process of vacuolization continues, until at the thirty-second day stage only the extreme oral end of the esophagus remains closed.

5. Both the closure and the reëstablishment of the lumen of the embryonic esophagus involve mechanical as well as growth processes, but are normal for a certain stage of the embryonic development. The closure is not largely dependent upon intrinsic cell division; and the fenestration process involves no tissue degeneration or resorption. The level of initial closure and the level of final perforation are approximately the same, namely, the laryngeal level of the esophagus.

6. In the process of vacuolization upon which the opening of the temporarily stenosed esophagus depends, the larger spherical vacuoles are drawn into irregular areolae as if through traction exerted from without. This traction no doubt inheres in the growing and expanding periphery of the esophagus. The esophagus now has a fenestrated appearance in section; its lumen is spanned by more or less delicate nucleated septa which may anastomose, giving to the whole the appearance of a wide-meshed syncytium. Ultimately the trabeculae are drawn into the lining epithelium, and their nuclei incorporated among the entodermal cells of the mucous lining.

7. The temporary atresia of the esophagus in the *Caretta* embryo would appear to be a device for the protection of the lung during its development against yolk material from the gut; which material could not be digested but would interfere with normal development of the lung.

8. This hypothesis can comprehend and correlate conditions in embryos of forms with meroblastic, homoblastic telolecithal, and alecithal eggs. Where yolk is very abundant as in the meroblastic eggs of fishes, reptiles and birds, the atresia is relatively extensive and of longer duration; in amphibia the closure is largely of the nature of a stenosis in which yolk globules are involved, probably in process of digestion while their forward progress is delayed by reason of the constricted lumen. In most mammals and in man such mechanism is functionally superfluous, and consequently absent except in slight and variable degree. As such it may persist or become accentuated, and produce congenital atresia or stenosis of the esophagus.

A more detailed description will appear in Publication No. 251 of the Carnegie Institution of Washington.

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STUDIES OF MAGNITUDES IN STAR CLUSTERS, V. FURTHER EVIDENCE OF THE ABSENCE OF SCATTERING OF LIGHT IN SPACE

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The interest and importance attached to an accurate quantitative knowledge of the scattering of light in space, for all studies of the extent and character of the stellar universe, has been commented upon by various writers, particularly by Professor Kapteyn. In the first communication of this series[1] the matter was discussed briefly and evidence was presented showing that interstellar space, at least in the direction of the Hercules cluster, is free of the kind of light absorption that modifies the color of the stars. The generalization of this